CHRONIC SUPPURATIVE OTITIS MEDIA AND SENSORINEURAL HEARING LOSS : IS THERE A CORRELATION?

Kamaljit Kaur[1], Nishi Sonkhya[2], A.S. Bapna[3]

ABSTRACT : *Traditionally, it has been accepted that chronic suppurative otitis media is associated with a breakdown in mechanical conduction of sound leading to conductive hearing loss. On the contrary, there are two schools of thought when it comes to the issue of cochlear involvement leading to sensorineural hearing loss (SNHL) in chronic suppurative otitis media. The present study was undertaken to find out whether a sensorineural component exists in hearing loss associated with chronic suppurative otitis media. A sample of 100 patients of unilateral chronic suppurative otitis media was selected for the study and their bone conduction thresholds were analyzed in relation to the duration of disease using audiometric data. A 24 per cent incidence of sensorineural hearing loss was found in this series, particularly involving the higher frequencies. Moreover, the incidence of sensorineural hearing loss progressively increased with the increase in duration of chronic suppurative otitis media.*

Key Words : *Sensorineural hearing loss (SNHL) and chronic suppurative otitis media (CSOM)*

INTRODUCTION

The role of chronic inflammatory disease of the middle ear as a cause of SNHL is still debatable. The hearing impairment in patients with CSOM has generally been observed to be an increase in air conduction thresholds but normal bone conduction thresholds. However, several investigators have reported that loss of cochlear function, and hence SNHL, does occur as a common sequel of CSOM.

Verhoeven (1961) and Thorburn (1965) made anecdotal observations of cochlear losses resulting from CSOM. Paparella et al (1984) hypothesized that CSOM can cause temporary threshold shifts or permanent threshold shifts by passage of inflammatory agents through the round window, which can spread apically and become measurable on routine audiometry. According to them, temporary threshold shifts occurred from serous labyrinthitis while permanent threshold shifts occurred from permanent dysfunction of the organ of Corti. They showed that the anatomical characteristics of the round window are such that it encourages the accumulation, stagnation and absorption of purulent secretions into the perilymph.

The round window membrane has 3 layers which together measure approximately 0.065 mm. The round window niche is approximately 1 mm in depth and 2 mm in diameter. There are essentially no ciliated cells in the region of the round window under normal circumstances. Pus can be pooled in the adjacent sinus tympani space, especially when the patient is in an upright position. Remnants of mesenchyme in the round window niche and sinus tympani are also slow to be resorbed. These factors encourage pus or infected tissue to be concentrated at the round window thereby encouraging absorption through the round window leading to chemical contamination of the perilymph.

In the light of this literature review, the purpose of this study was to evaluate the incidence of SNHL in CSOM and to study the correlation of duration of disease with the incidence of SNHL.

MATERIALS AND METHODS

The records of 100 consecutive patients with unilateral CSOM attending the Department of ENT in SMS Medical College and Hospital, Jaipur between Jan-June 2001 were evaluated. Only patients aged 11-50 years were included in the study. Patients younger than age 11 were excluded to eliminate the possibility of inaccuracies of audiologic testing in children. Patients older than 50 years of age were excluded because of the small number of patients in this age group and the increased incidence of presbyacusis in this age group. The following patients were also excluded from the study : (i) previous otologic surgery (ii) history of familial hearing loss (iii) previous exposure to ototoxic drugs (iv) a positive

[1]Senior Registrar, [2]Assistant Professor, [3] Professor & Head , Department of ENT, SMS Medical College & Hospital, Jaipur

Table – I : Demographic profile of 100 CSOM patients

Sex		Age group (years)	No. of patients
Male	61	11 - 20	37
Female	39	21 - 30	29
Total :	100	31 - 40	24
		41 - 50	10

Table II : Bone conduction thresholds in patients of CSOM having SNHL

S. No.	Age (Years)	Duration of disease (Years)	Bone conduction thresholds (Decibels)		
			1K	2K	4K
1.	28	18	20	25	30
2.	14	10	20	30	40
3.	34	24	30	40	50
4.	27	19	20	25	30
5.	32	20	30	40	60
6.	30	15	20	25	30
7.	41	26	20	40	60
8.	48	>30	30	50	60
9.	25	17	30	35	40
10.	21	11	20	30	40
11.	42	24	30	50	60
12.	18	8	20	30	40
13.	37	28	30	35	40
14.	35	24	20	35	50
15.	17	5	20	25	30
16.	22	12	20	30	40
17.	19	14	20	30	40
18.	33	22	20	25	30
19.	18	4	20	30	40
20.	24	17	20	25	30
21.	16	8	20	25	30
22.	25	16	20	25	30
23.	14	3	20	30	40
24.	20	15	20	25	30

fistula test (v) frank labyrinthitis (vi) history of habitual exposure to noise (vii) history of head trauma (viii) any other apparent factors which could result in SNHL. Sex, age, duration of disease and audiometric data were recorded for all the patients and the incidence of bone conduction loss was compared to the duration of disease. For each patient sampled, bone conduction frequencies were measured at 1K, 2K and 4K. Patients with unilateral CSOM only were included so as to indicate infection as a cause, rather than other possible etiologies in causing SNHL, the normal ear thus serving as a control.

Table – III Correlation of duration of disease with incidence of SNHL

Duration of disease (Years)	No. of Cases	Incidence of SNHL
< 5	22	3 (13.64%)
6 - 10	16	3 (18.75%)
11 - 15	21	5 (25%)
16 - 20	19	6 (31.58%)
21 - 25	13	4 (30.77%)
26 - 30	6	2 (33.33%)
> 30	3	1 (33.33%)

OBSERVATIONS AND RESULTS

The demographic profile of 100 patients of CSOM showed that the M : F ratio was 61 : 39 and 66% of the patients belonged to the 11-30 year age group (Table I).

The bone conduction thresholds of patients exhibiting SNHL at 1K, 2K and 4K were compiled to show the range of variation in the bone conduction thresholds (Table II).
It was also observed that as the duration of CSOM increased, the incidence of SNHL also progressively increased (Table III).

DISCUSSION

CSOM is one of the most common conditions encountered by the otologist in his routine day to day practice. CSOM has been conventionally described in terms of loss in the conductive component of hearing. The present study was undertaken to evaluate the presence of a sensorineural element in hearing loss associated with CSOM.

In this series, it has been our clinical impression that SNHL does occur in CSOM with particular involvement of higher frequencies. An incidence of 24% of SNHL was detected in 100 cases of unilateral CSOM. Although the work of many eminent investigators has supported this view, there are others who deny an association between CSOM and SNHL.

Gardenghi (1955) found that 22 (44%) of his 50 patients with CSOM had cochlear hearing loss while Bluvshtein (1963) reported that 37.5% of his patients with CSOM had some loss of cochlear function. Paparella et al (1970) reported an increased incidence of SNHL in a study of 232 patients. English et al (1973) also reported the occurrence

of SNHL in CSOM. Papastavros and Varlejides (1986) in their study of 66 cases of CSOM, found the presence of SNHL with a reversible and a permanent component in the diseased ears, the reversible component involving the higher frequencies and the permanent component being equally present in the whole range of the tested frequencies. Blakley & Kim (1998) studied 123 cases of unilateral CSOM and reported a highly significant correlation between SNHL and CSOM. Dumich et al (1983) reviewed 200 consecutive patients of unilateral CSOM and concluded that clinically significant SNHL is uncommon in patients with CSOM. Kaplan et al (1996) studied 127 diseased ears in children and reported that CSOM has little effect on the cochlear function.

When the duration of disease was compared with the incidence of SNHL, a progressively increasing incidence of SNHL was found as the duration of disease increased. The incidence of SNHL was found to be 13.64% when the duration of disease was < 5 year and progressively rose to 33.33% when the duration of disease was > 26 years. Similar correlation between duration of disease and SNHL has been reported by Paparella et al (1970), English et al (1973), Cusimano et al (1989) and Kholmatov (2001). However, Noordzig et al (1995) and MacAndie et al (1999) did not find a correlation between SNHL and duration of CSOM.

Huizig (1964) acknowledged the presence of bone conduction loss in CSOM, stating that while the loss is generally considered to be due to cochlear damage resulting from inflammation, more often the lowered bone conduction thresholds have a middle ear origin which he described both as "Middle ear bone conduction loss" and a "pseudoperceptive loss". He also stated that treatment of the middle ear disorders may improve bone conduction thresholds and allow a more accurate measurement of cochlear function.

The mechanism of occurrence of cochlear damage, and hence SNHL, in CSOM has been studied by temporal bone studies and animal models. Rauch (1965) has demonstrated an increase in LDH and especially malic dehydrogenase in the perilymph of the guinea pig following experimentally induced middle ear infection. Paparella et al (1972) studied the role of round window in transmitting inflammation from the middle ear to the labyrinth. Temporal bone studies demonstrated slight hyperplasia and metaplasia of the outer layer of round window in all specimens. Gross changes seen in round window consisted of dilatation of the vessels within the fibrous layer, thickening of the membrane, infiltration of inflammatory cells, marked metaplasia or hyperplasia, or cystic changes. The most significant changes were found in the perilymph of the scala tympani near the round window membrane, although perilymph changes in other areas were occasionally found. These alterations consisted of either serofibrinous precipitates or inflammatory cells in the perilymph or both.

Goycoolea et al (1980) conducted a longitudinal sequential study of oval and round window changes in otitis media in cat using eustachian tube obstruction and concluded that the gradual histopathologic changes seen in the round window membrane plus the serofibrinous precipitates were clearly suggestive of an active reaction and led to a change in the permeability of the round window membrane. The semipermeable nature of this membrane has also been demonstrated by observations of passage of sodium-22 and horseradish peroxidase through the round window membrane into the labyrinth.

Guo et al (1994) studied the effect of endotoxic damage to the stria vascularis and concluded that lipopolysaccharide induced strial ototoxicity produced ion imbalance, causing changes in endolymph composition and energy failure in the organ of Corti and hence explaining the pathogenesis of SNHL in CSOM. Engel et al (1998) studied the passage of radio-iodinated streptolysin-O and albumin through the round window membrane and proposed that the passage of noxious macromolecules, such as proteases, from a purulent middle ear effusion may be facilitated by pore-forming toxins, resulting in cochlear damage and SNHL.

In the light of this literature review, there is a need for the continual assessment of sensorineural function in patients with CSOM. When SNHL develops, active surgical or medical intervention should be considered if not already done. Too many physicians in medicine consider CSOM to be an innocuous process unless an obvious complication develops.

CONCLUSION

Audiometric data of 100 cases of unilateral CSOM were evaluated using the other (normal) ear as a control. A 24% incidence of SNHL in CSOM was found in this study and it was also observed that the incidence of SNHL increased with the increasing duration of disease. More audiologic studies in patients with CSOM are needed to evaluate the

sensorineural component of hearing loss It would be interesting if such studies included the entire upper frequency spectrum since it is possible that very high frequency losses occur which are not perceptible to the patient and are not measured by routine audiometry A high frequency audiometry test (i e 10,000 Hz to 20,000 Hz) is advisible in all patients of CSOM

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Address for Correspondence :
Dr Nishi Sonkhya,
7/251, Vidhydhar Nagar,
Jaipur - 302012 (Rajasthan)